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THE USES OF AN ARTIFICIAL EYE-SHELL FOR OPERATIVE AND OTHER PURPOSES.

BY THOMAS R. POOLEY, M. D.,

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1. *Symblepharon.*—"Numerous are the expedients which have been resorted to for the cure of this affection, but they all have for their object the mechanical separation of the lid and eye-ball until the surfaces cicatrise. Some surgeons endeavor to effect this by simply disecting the adherent lid away from the eye-ball, and then tearing the wound daily for a certain period, until it no longer unites; others, again, try to keep the surfaces separate by interposing foreign bodies, such as sealing-wax, glass shields, or similar substances. It is always found, however, that nature frustrates any attempt to remedy the deformity by such expedients, for they are based upon the supposition that a delicate physiological action can be replaced by a mechanical contrivance, whereas whenever such surfaces rest upon each other they ultimately adhere together."

The above quotation is taken from Wolfe, Diseases and Injuries of the Eye, and it must be admitted is only the reflection of an opinion which is almost universal as to the futility of keeping separate the adherent surfaces after dividing a symblepharon by any mechanical contrivance.

Nevertheless the success which this writer has obtained by just such a procedure seems to warrant the report of the following case:

Miss K., æt. 46, came to the New Amsterdam Eye and Ear Hospital on Dec. 28, 1888. In both eyes were the upper lids adherent to the globe.

In the left where the adherence of the lid was almost throughout its entire extent the cornea was leucomatous, and there was no possibility of restoring any vision.

In the right, however, in which the middle third of the lid was adherent to the eye-ball, the cornea although pannous, was sufficiently clear to permit of vision sufficient for the patient to see her way about.

Besides the adherence of the lid there was also entropion, so that the lashes swept the cornea. The whole condition was probably due to a neglected or badly treated trachoma. Instead of resorting to an operation for transplantation of the symblepharon as recommended by Teal, Wolfe and others, I determined to try keeping the opposing surfaces separated by use of an artificial glass shield, which had lately been brought to my notice, shaped exactly like an artificial eye, but of transparent glass, carefully smoothed and polished, so as not to irirate the eye over which it is inserted.

These shells were brought to my notice by M. Brière, formerly an associate of Boissonneau, of Paris, who told me that they could be worn over a perfectly healthy cornea, as had been proved by himself.

The method of operating was as follows: The symblepharon, which was found to consist of two bands, was freely dissected from the eye-ball, so that the lid was easily lifted from the globe, the cul de sac being fully sutured. Knapp's entropion clamp was then introduced and Snellens' operation for entropion made.

The glass-shell, shaped exactly like an artificial eye, was then introduced, care being observed to use one sufficiently large, so that the low surfaces were kept apart. Care was taken to have it thoroughly clean, by keeping it for some time in a solution of bichloride of mercury, 1-5000.

The glass eye which fitted exactly like an artificial eye was then introduced and tolerated by the patient with but little in-

convenience and no pain. It was allowed to remain for 24 hours before its removal and caused so little discomfort that it did not interfere with the patient's sleeping, and she could see through the glass to find her way about. The shell was allowed to remain for a week, only removing it 3 times a day for the purpose of cleansing the eye and the artificial shell. At the expiration of this time the surface on the lid was so nearly cicatrised that it was only found necessary to wear the shell every alternate hour and at last only during the day, it being left out at night. The patient remained in the hospital for four weeks and was then discharged—both surfaces having entirely cicatrised. The lid was entirely free from the globe with the exception of a single very narrow band of adhesion which could, however, only be seen when the lid was forcibly raised so as to expose the bottom of the cul-de-sac, and which was no doubt caused by the fact that the glass-shell was hardly large enough to prevent the contact of the surfaces at this place. The depth of cul-de-sac, however, obtained was quite as good as that usually resulting from the different methods of transplantation, and the movements of the eye were not restrained in any direction. Her sight which had been only equal to quantative perception of light was $^{20}/_{cc}$.

Whether this method will succeed in every instance or not must be decided by further experience. There can be no doubt that the foreign body, the glass-shell, was the better born in this case on account of the condition of the cornea, for, as is well known, all sorts of operative procedures are well borne in cases of pannus of the cornea.

II. I would suggest that this shell which can be readily tolerated over the natural healthy cornea, may prove of use in preventing the infection of the healthy eye in cases of blennorrhœa, and at the same time prove more comfortable to the patient than the methods now in use of excluding the healthy eye by permitting the patient the use of vision.

These shells are manufactured by M. Brière of No. 838 2nd Av., New York.

RUPTURE OF THE CHOROID.

BY DAVID COGGIN, M. D., SALEM, MASS.

Rupture of the choroid being of infrequent occurrence the brief history of two cases of this accident may not be without interest.

1. 1st December, 1884.—Fred. A., æt. 20, was seen in consultation. Three days before, while playing polo in a rink, he was knocked down by a polo-stick, cutting the skin of his left eye-lid and also that near the outer canthus so that sutures were called for.

He was in bed, as he had suffered from nausea and was still feeling badly. Eye not tender. Some ecchymosis of lids. Pupil widely dilated and nearly hidden by a thin layer of blood which extended to the nasal periphery of the anterior chamber and whence it was then thought the hæmorrhage had come. Tn. V.=Fingers at 15 centimetres. On the following day, at my office, he saw fingers at 1 m. ($^4/_{IV}$ *o. d.*) Unable to converge, *o. s.* and slight ptosis. Blood absorbed from the anterior chamber but no view of fundus.

9th Dec. Through hazy media, made out what seemed like separation of the retina in the region of the macula.

16th. Now can converge eye. Pupil continues dilated. Central scotoma. What was taken for retinal detachment now proved to be an extensive rupture of the choroid—the sclera being visible—on the temporal side of the disc and resembling an inverted Y.

7th April, 1885. V. unchanged. The place of rupture filled with gray-black pigment, the two prolongations below appearing as delicate cicatricial lines. The patient was not seen again as he fell a victim to pneumonia the succeeding year.

2. James, a Salem boy æt. 11 years, was hit in his right eye by a stone thrown by a street Arab just as he was brought to me, the 10th of October, 1883. There was a large but superficial central abrasion of the cornea and a bit of stone was impinged in its lower border which was at once removed.

V. quantitative. Pupil dilated. Ordered atropine salve, a band and the like. The next day V=$^{4}/_{LX}$. 22d Oct. V=$^{4}/_{XXXVI}$. Sees a 'notch' when he looks at any object. Media still too cloudy to allow a view of the fundus. Some pain and ciliary redness.

A month later, a semilunar rupture of the choroid was seen on the temporal side of the disc, extending above and below it and distant about the diameter of the disc from the disc-border.

A dark round deposit, like the remains of a clot, in its centre. 3rd Dec. V. $^{4}/_{XXIV}$. Rent contracting over exposed sclera. Patient says he over-reaches when handling his toys. Occasional pain goes from head to eye. Pupil yet slightly dilated.

11th Feb., 1884. V.=$^{4}/_{XVIII}$. 'Notch' less trying. Site of tear in choroid now recognized only by its light color. Some letters of Sn. 1 made out. When next examined, in May, 1887, no view of the fundus was obtained owing to the increase of the zonular cataract, which had first been seen in its incipient stage, five years earlier.

Zonular Cataract.

J. O., æt. 10, a promising boy, belonging to a well-known Massachusetts family, was brought to me in September, 1882, because it was thought he could not see well with his right eye.

V.—$^{4}/_{VI}$ *o. u.* xl. D.=$^{4}/_{IV}$ *o. s.* No glass helped o. d. xl.(+1.5) 5=$^{50}/_{L}$ *o. u.* H. by mirror. Fundus hazy owing to slight, disc-like opacities in both lenses and suggestive of zonular cataract. The retinal vessels, etc., could be fairly seen though less distinctly than through the periphery of the lenses.

An aunt had a 'cataract' and became blind at twelve.

His sister has irregular as., and his grandfather had 'peculiar sight.' In the ensuing July, atropine was used and V.=$^{4}/_{XXIV}$ *o.s.* $^{4}/_{IX}$ *o.d.*—no glass helping. Opacity in lenses increasing. In October, as stated before, he suffered a rupture of the choroid (o. d.) 4th Jan., 1884, V.=$^{4}/_{VI}$ *o. s.* $^{4}/_{XXIV}$ *o. d.* 22d May, 1887, V.=$^{4}/_{XII}$ *o. s.* and $^{15}/_{L}$.

$^{4}/_{LX}$ *o. d.* and reads Sn. l. Cataracts more dense—periphery still transparent. In such a case, where the trouble has steadily increased, it certainly seems as if any thing short of the removal of the lenses would be of only temporary benefit.

The following case is hardly analogous but it may be worth relating. 22d January, 1887. Wm Fitzgibbons, æt. 39, N. B.—He sought advice at the Salem Hospital, because of life-long 'near-sightedness' V.=$^{4}/_{XXIV}$ *o.u.* After atropine it was $^{4}/_{XVIII}$ *o.d.* only. Unable to read fine type. By the ophthalmoscope, typical zonular cataract *o. u.* Glasses of no help. 29th Jan.—Ether. Iridectomy, in and down, both eyes. 4th Feb. V.= $^{4}/_{XII}$ *o. u.* and Sn. l. readily. ('Never seen so well before.')

A NEW OPTOMETER.

BY ELMER STARR., M.D.

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This optometer is constructed of two tubes 8 cm. long, one of which telescopes within the other. Each tube is fitted with a lens at one end, the outer tube carrying a —16.D., the inner one a +16.D., so that when the tubes are closed the plus glass exactly neutralizes the effect of the minus glass and the combination equals 0. In using this optometer the eye to be tested looks through the instrument from the opposite end to the one carrying the lenses, and examines the test type ordinarily used in testing vision, placed twenty feet away. It will be observed that by this arrangement the eye is kept at a distance of 8 cm.—the length of the tube—from the plus lens of the instrument; by this means the size of the letters and their apparent distance remain unaltered and appear the same as when a single lens which the combination represents is used. When the instrument is closed the effect is the same as a plain glass, and the combination stands at 0, so that the test types appear the same as when examined without a glass. When the tubes are extended the result is a plus combination, whose power gradually increases as the tubes are extended, passing from 0 up to +16.D. when the tubes are fully extended and the lenses most widely separated. So that by examining the test types through this instrument an error in refraction in the eye requiring a plus glass for its correction can readily be determined.

For the minus combination the cell holding a —16.D. lens is slipped off into the proximal end of the inner tube. Now

when the instrument is extended this —16.D. just neutralizes the effect of the other two lenses, and the result is again a plain glass or o. Then as the instrument is closed the result equals a minus glass whose power gradually increases up to —16.D. when the tubes are closed.

The tubes are worked with rack and pinion, and are marked with a scale, so that the result can be read off immediately after the correction has been found. The circumference of the tube is also marked, at its end, with the degrees of a circle, for use when correcting astigmatism. When used for this work a disk having a stenopaic slit is fitted to the end of the instrument so that the slit can be inclined at any angle.

The optical principles of this instrument are these: The —16.D. lens with which the outer tube is fitted forms a virtual image of the test types examined, about 6.25 cm. in front of the instrument; that is, just at the focus of the +16.D. lens of the inner tube, when the tubes are closed and the lenses in contact. So that rays of light from the test types, after passing through this refracting system, leave the plus glasses parallel, or just as if they had come from the type without passing through any refracting system. When the glasses are separated by extending the tubes the strength of the refracting plus combination may be determined by the following equation:[1] $\frac{p-f}{f} = \frac{f}{p'-f}$

Suypose the tubes are extended until the glasses are separated by 1 cm. Then the plus glass will be 6.25+1=7.25 cm. from the virtual image. Replacing the letters with figures in the equation we have: $\frac{7.25-6.25}{6.25} = \frac{6.25}{p'-6.25}$ $p'=45.31$.

That is, the effect of separating the lenses of the instrument

[1] f denotes the focal length of the plus lens, p the distance of the image (object) from the plus lens, and p′ the focal length of the resulting combinatio n.

1 cm. equals a plus glass of 45.31 cm. focus, or about +2.20 D. But in using a plus glass to examine test types twenty feet away, or at any distance twice the focal length of the glass used, the effect or power of the glass increases with its distance from the eye. Now the glass in the tube of the optometer is 6.25 cm. in front of the eye looking through the instrument. Therefore, although the effect of separating the glasses 1 cm. equals a lens whose focus falls 45.31 cm. behind it. Yet this lens is situated 6.25 cm. in front of the eye, which brings its focus 6.25 cm. nearer the eye. That is, it has the same effect upon the eyes as a lens of 45.31—6.25= 39.06 cm. focus =2.56 D. Hence, extending the tubes of the optometer 1 cm. gives a +2.56 D. In the same way it can be shown that the effect of extending the instrument fully, *i. e.*, 6.25 cm., equals +16 D. From this it is readily seen how any minus combination may be obtained by placing a —16 D. in the end of the tube, as already explained.

Similar calculations will give the effect when examining test letters for the near point. In this case, a different distance being used, a different result is obtained; so that the tubes are marked with a second scale for use when testing vision for near work.

ADVANCEMENT, WITH BUT A SINGLE SUTURE.

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I have for the past two years, with most gratifying results, practiced in all my cases of advancement a method which may be briefly described as follows:

The lids being held apart in the usual way, a long horizontal incision is made through the conjunctiva over the tendon of the muscle to be advanced, and carried quite up to the cornea. The conjunctiva is dissected up well around about the insertion of the muscle, care being taken not to disturb it in the immediate vicinity of the cornea. The tendon is caught up on an ordinary strabismus hook, which is slid back and forth a time or two, to free the muscle and tendon from any attachments to surrounding parts, then given to an assistant to hold. Next a double-armed thread (No. 3, black) is taken —two delicate needles, curved on the flat from the point back half way to the eye on either end of the thread. One needle is passed downward through the tendon near its upper edge (or, even through the belly of the muscle, if the squint be excessive), and the other also, downward, though near the lower edge, thus throwing a stitch across the central portion of the tendon (or muscle) on its outer aspect. (See Fig. 1). An important item being always to keep the upper and the lowar needle quite distinct and separate for obvious reasons. The next step is to cut the muscle at the distance of about two millimeters from the stitch—on the corneal side, of course—and to give the thread in charge of an assistant. (Though I have

done the operation alone.) Now, pick up the stump of the tendon and cut it off close to the sclerotic, which procedure adds greatly in the way of an elegant result. After this, the upper needle is to be introduced beneath the conjunctiva, stitching it along through the episcleral tissue, and bringing it out near the cornea at a point opposite to its vertical diameter, and the same with the other needle below the cornea. Then the upper needle (or the lower, it is immaterial) is carried back and the point passed under the loop of thread lying

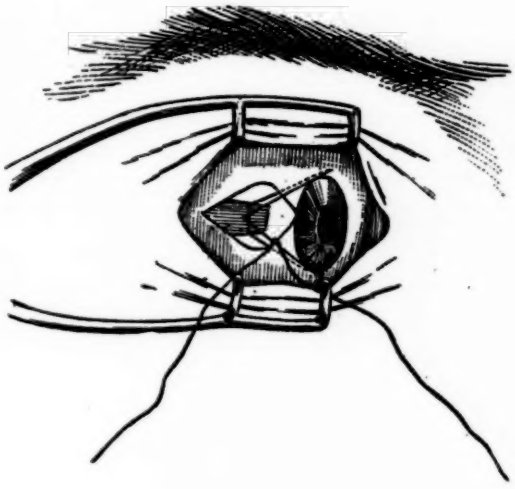

FIG. 1.

across the muscle, introducing it from behind, even allowing it to pick up a slight hold on the muscle on its way. After drawing the thread well through, both needles may be removed and the suture knotted. This stage does not differ essentially from other forms of advancement operations.

As in other operations, one must regulate the degree of tension applied in trying the thread, by the amount of effect desired; and it is most essential, particularly where no tenotomy, or only a partial one, of the counteracting muscle has

been made to have the globe rotated in the direction of the muscle to be brought forward, otherwise the suture is liable to cut or tear.

I greatly prefer not to have the patient anæsthetized, and it is really remarkable, that a large majority of the patients suffer absolutely no pain when submitting to this operation under cocaine alone.

I do not claim for this method like the man did for his Indian medicine, that one of the chief recommendations is its never failing efficacy, but I do think it has its advantages, the greatest of which is the inevitable advancement of the muscle in the direct line of its axis, as well as of its natural action. In this it would seem to be more exact than any other pulley operation ever brought to my notice; the very nature of the suture's arrangement precluding the possibility of fixing the muscle awry, and there is nothing to prevent the cut end of the muscle from coming forward, if necessary, quite to the margin of the cornea.

The operation is, moreover, very simple, and when the time arrives for the removal of the suture but a single snip of the scissors is required, when the thread may be easily withdrawn with forceps. I never close the conjunctival opening by sutures—merely arrange it, as well as possible, over the place with the aid of toilet forceps.

If it is necessary to combine a tenotomy of the opposing muscle with the advancement, I always do the latter first.

ON PTERYGIUM.[1]

BY ADOLF ALT, M.D.

GENTLEMEN.—The subject I want to draw your attention to may seem a very small one, yet as in the building of a house, every particle counts, and is important, so it is in medicine. I have selected this subject, *pterygium*, first, because it will not take much of your valuable time; and second, because in my connection with the Missouri Pacific Railroad I have come so much more frequently in contact with this affection than ever before. I cannot give the percentage in which the firemen, brakesmen and conductors of a railroad are subject to the growth of a pterygium, but that it is a very prevalent affection among them I need probably not tell you, and almost every-one of you may have to deal with it.

Pterygium is a growth of a more or less triangular shape, with the base in the conjunctiva bulbi, while its apex encroaches more or less upon the cornea. This triangular growth may be found to lie in all the different meridians of the eyeball; it lies generally, however, in the direction of one of the recti muscles, and its seat of predelection is that part of the eyeball which during waking hours lies exposed behind the palpebral fissure, that is, the horizontal meridian; but by far the greater number of pterygia lie on the nasal side of the eyeball, over the internal rectus muscle.

This triangular growth may reach only the edge of the cornea, or it may gradually grow toward the centre of this membrane, and even grow beyond it. In rare cases two such

[1]Read before the National Association of Railway Surgeons, held at St. Louis, May, 1889.

pterygia coming from diametrically opposite directions may meet and unite with each other.

While small pterygia are usually flat and but little elevated over the surrounding conjunctiva and the level of the cornea, larger ones may represent a thick, vascular, fleshy growth which rises over conjunctiva and cornea, the edges of which lap over the neighboring conjunctiva so that a probe can be entered for quite a distance beneath them. In internal pterygium the lachrymal caruncle is not unfrequently stretched and pulled toward the cornea, so as to lie upon the bulbar conjunctiva instead of its normal position.

The nature of the pterygium has been studied frequently under the microscope. It consists essentially and purely of conjunctival tissue. In order to explain this curious hypertrophy two theories have been advanced in former years. According to the one, ptergium is a polypoid growth of the conjunctiva, which being pressed on the cornea by the lids becomes agglutinated to this membrane. Arlt of Vienna, who originated the second theory, thought that a marginal ulcer of the cornea becomes covered by an overlapping fold of the adjacent swollen conjunctiva, and that the growth of the pterygium is due to a continued ulceration of the cornea at the apex of this conjunctival growth.

Neither of these theories fully explains the conditions. If every marginal ulcer of the cornea was able to produce a pterygium, pterygia would be much more common than they are. There must be something more than a common marginal ulcer.

Some 12 years ago I had, as the first, the opportunity to examine a whole eyeball with a pterygium histologically, and my publication was soon followed by one by Goldzieher of Buda-Pesth. These two cases seem to be the only ones so examined to this day. As new they brought to our knowledge, that epithelium is found under the pterygium and lying upon the sclerotical and corneo-scleral tissue. This proves as certain that a fold of conjunctiva must have been glued to the underlying tissue. This epithelium undergoes slow retrogres-

sive metamorphoses, and I have twice had occasion to see a large cyst formed under a pterygium containing colloid fluid, probably due to the metamorphosis of this incarcerated epithelium. Such observations prove beyond a doubt that a fold of conjunctiva is glued to the cornea. The fact that the lachrymal caruncle is found lying upon the bulbar conjunctiva shows, that during the further growth of the tumor a very considerable traction must again and again be exerted upon the conjunctiva at the base of the original fold. Where this force and the continued stimulus come from is still an open question.

In a recent number of THE AMERICAN JOURNAL OF OPHTHALMOLOGY Dr. Theobald, of Baltimore published an article of the pathogenesis of pterygium in which he claimed that on account of the intimate connection between the vascular system of the recti muscles and that of the conjunctiva in the neighborhood of the corneal border these muscles, by influencing the blood supply of the overlying conjunctiva do, in fact, play a most important role in determining the formation of a pterygium. Further on he comes to the conclusion, that the internal recti muscles would exert this influence by far more decidedly than any of the other recti, and that thus we have a satisfactory explanation of the fact that pterygium occurs in so large a majority of the cases to the nasal side of the cornea.

This theory sounds quite plausible. Yet, if true for the internal ptergia, how would Theobald have us understand the formation of pterygia in the direction of other recti, or those lying in other meridians? Moreover, if this theory for the explanation of how internal pterygium may be formed were correct, why should it be most common among those classes of people whose internal recti muscles are perhaps least strained? Should we not expect internal ptergia in the majority of eyes that are daily and continually used for near-work? Yet just in these classes they are almost totally wanting.

A much better, and perhaps the true explanation, at least for the prevalence of internal pterygia, was given by Dr. Young

of Burlington, Iowa (see this journal, volume IV, No. 10, page 302). He says: To shut an eye to irritants and yet keep it open sufficiently to see any work ahead the orbicularis necessarily contracts irregularly. Extremely to the temporal side, moderately through the rest of its course. The result is that in the great majority of people the eye is well protected except over the centre of the cornea, and a small strip to the nasal side of the cornea.

This explanation is, I think, a very good one as far as it goes, that is, as long as we have to deal with internal pterygia only—but it will in no way apply to pterygia corresponding to the vertical meridian and others.

The easiest way out of this difficulty is the assumption of a microbe which falls into, or even causes, a small ulceration of the cornea, which then is covered by a small fold of conjunctiva. As the microbe grows into the cornea, and nature tries to heal the ulcer, the conjunctiva is dragged along. This theory, or a similar one, was propounded by Poncet, without histological proof, as far as I know. In the specimen of a whole eye I had occasion to examine, the apex of the pterygium entered the corneal tissue like a wedge, and the corneal lamellæ nearest to it were filled with small granules, for which I then had no explanation. Perhaps they were such microbes. I think, therefore, that Arlt's theory, combined with Poncet's, explains the formation of pterygia still best; that is, that for the formation of a pterygium we must first have a loss of substance in the corneal margin; second, a microbe which grows slowly into the corneal tissue; and third, a small fold of conjunctiva, dragged on the cornea by the continued progress of microscopical death and attempted repair in the corneal tissue.

Such premises are easiest brought into existence in the life-work of men who are continually exposed to wind and weather, to heat and the flying of small foreign bodies; and the classes to whom this would apply are just the ones most frequently attacked by pterygia, namely: farmers, seamen, blacksmiths, firemen, stokers, glassblowers, and last but not least, railroad employees.

If it was possible to prevent the formation of pterygia, a great boon would be conferred upon these people. Two things might suggest themselves to serve this end. First, protective spectacles. They would, however, be of little use, unless they fit closely to the face, which is disadvantageous in other directions. Spectacles which do not fit closely act, in my opinion, rather like a chimney by means of which foreign bodies are drawn into the eye. Protective spectacles, therefore, will not do to prevent pterygium, even if it should be possible to get these people to wear them.

The other preventative might be the use of an antiseptic wash, night and morning. I am satisfied, that this means has in my experience already done some good, and is capable of doing a great deal more, I think, if practiced more generally.

But, how shall we deal with a pterygium when the patient presents himself? The only lasting remedy is the removal of the growth. The number of ways in which this is done is large, and I shall not try to bore you by reviewing them all. The most well known and most frequently practised operations are the following:

1. The simple excision. This operation which is probably the oldest one, consists of a careful dissection of the apex of the pterygium from the cornea, and the excision of a rhomboid piece of conjunctiva. The wound over the sclerotic may or may not be closed by stitching the conjunctiva. This operation prevented a relapse, as a rule, in small pterygia only. For larger ones it was seldom successful.

2. Transplantation of the pterygium into the retrotarsal fold. This operation was introduced by Desmarres, who, after dissecting the pterygium, made a cut in the retro-tarsal fold of the conjunctiva, going up or downward from the base of the pterygium. The whole growth was then stitched into the resulting gap, after the manner of a twisted flap. Knapp, not satisfied with the resulting asymetrical swelling thus produced in the lower or upper cul-de-sac, improved this method by cutting the pterygium after the dissection in halves, and by trans-

planting one-half downward and the other upward. The results of these operations were good, as a rule.

3. An operation whose author I do not know. Its first step is the dissection of the pterygium. Then the conjunctiva is undermined at the base of the growth and a thread armed with two needles being passed through the apex of the ptergium, the latter is turned under the undermined conjunctiva, rolled up, as it were, and there fastened by the sutures. An ugly looking swelling at first results from this procedure, which, however, gradeally disappears as the ptergium atrophies. The final results are mostly good.

4. Prince of Jacksonville once accidentally tore a pteryggium off from the cornea, and the result, according to his description, was so excellent that he seems to have since adopted this method of evulsion for all pterygia. I have never tried it, and never shall for obvious reasons.

All of these methods aim at a successful removal of the growth with the prevention of a relapse, and at rendering the part of the cornea on which the growth had been lying as transparent as possible.

Starting from the idea that microbes play an important role in the formation and growth of a pterygium, I have for several years removed all pterygia in the manner which I shall now detail.

After having carefully disinfected the conjunctival sac, the eyelashes, lids and eyebrows by means of a sublimate solution of one in 2,500, and having the eye well concainized, I grasp the pterygium with toothed forceps, and gently pulling it away from the eyeball, dissect it off from the cornea as neatly as I possibly can. I prefer for this part of the operation a bent lance-shaped knife. Then I make the rhomboid excision, detailed before, with scissors. Next the bulbar conjunctiva is well undermined up and downward and brought together by one or two sutures. Then the uncovered portion of the sclero-corneal tissue and the loss of substance on the cornea are *cauterized with pure carbolic acid.* This procedure is followed by the instillation of a solution of sublimate of one in

from 3,000 to 5,000 every two hours for some days, then less frequently. I have done this during the last year on sixteen railroad men alone. The results were all that could be wished for, that is, not only has no relapse occurred in these cases, but the cornea has cleared up nicely, and, in some severe cases, wonderfully.

I instruct the patients after I have removed the sutures, which I do on the fourth or fifth day, to continue using a sublimate solution of one in 5,000, nights and mornings, and I can recommend this method. It simplifies the operation and gives better results than the newer methods of transplantation. The method is simply the old careful excision with antiseptic measures before and after the removal added to it.

TRANSLATION.

EXPERIMENTAL RESEARCHES CONCERNING THE ANTISEPTIC AND GERMICIDAL ACTION OF CREOLIN.

BY E. VAN ERMENGEM.

(*Bulletin de l' Academie royale de médicine de Belgique.*)

This excellent critical and exhaustive article ends with the following conclusions drawn from a number of series of experiments.

"The sample of creolin which was furnished me by the firm of Pearson & Co., of Hamburg, has proved itself to be an antiseptic of the first class, decidedly superior to phenic acid, and comparable only to the bichloride of mercury.

Although it may happen in practice that this action is retarded or even considerably reduced by the presence of serous and albuminous substances, the 5 per cent solutions of creolin still merit the preference of the surgeon, on account of their less irritating action, than that of the acidulated solutions of the bichloride of mercury or of phenic acid, and on account of the absence of any poisonous effect.

Its faculty to kill the spores does not surpass that of phenic acid when tartaric acid is added, yet it almost equals it.

The usefulness of 5 per cent solutions of creolin for disinfecting purposes is due to their very energetic germicidal action with regard to many of the most important pathogenic microbes: namely spirillæ, cholera bacilli, typhoid fever bacilli, strep-

tococcus of erysipelas (puerperal fever), staphylococcus pyogenes. If their innocuousness in man should become confirmed, we could not refuse to give these solutions the first position among all the disinfecting fluids, because their application could fearlessly be left to the nurses and to the even most ignorant public, especially in times of epidemics and for the antisepsis during confinement.

In fact, it seems that creolin combines all the qualities which the hygienist must ask from a disinfectant which should be useful in general practice. It acts as a germicide, and its action upon infecting substances, whatever their origin, or under whatever form they present themselves, is most certain. Its action is rapid and it can be used profusely without fear of poisoning, and without spoiling the object (linen, bedding, etc.,) which are submitted to its action.

It is, furthermore, an excellent desodorant, a quality which is by no means without its value. Finally the price is low, and it can in consequence be used largely for hygienic purposes of the most varying character.

All experiments should be heartily encouraged which are made for the purpose of giving the precise knowledge of its composition, of assuring its invariability, of rendering it still more active, and of eliminating the inert substances which it now contains.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

THURSDAY, MARCH 14, 1889.

J. W. HULKE, F.R.C.S., F.R.S., President, in the Chair.

Color Blindness and Color Perception.—Dr. Edridge-Green read a paper in which, after mentioning in detail the objections, which seemed to him of greatest importance, to the Young-Helmholtz and the Hering theories of color vision, he explained at length his own views and the theory which he had built up as a result of numerous experimental examinations of persons with normal and defective color perception. He held that the color perception centre of every individual was able to appreciate a certain number of units of color, these units corresponding more or less closely to the bands of the solar spectrum. The average number of units was six, namely, red, orange, yellow, green, blue and violet; but persons of unusually good color perception possessed a seventh, namely, indigo or dark blue, which was placed between the blue and the violet. In people with color perception below the average, one or more units of color would be wanting; orange was the first to disappear, and it was replaced by a widening of the red and yellow bands. Such an individual would belong to the five-unit class. Blue was the next band to disappear, the violet then extending to the normal blue-green junction. The next band to fail was the yellow, the red then reaching to the green. The green and red then became as one band, and so the units were reduced to two, the violet still remaining; in total color-blindness these two were replaced by a neutral band. Dr. Edridge-Green gave the following "laws of color perception," which he deduced from the facts obtained in his investigations: 1. An

individual can have no conception of a color which does not form one of his psycho-physical color units, or a very apparent modification of one of them. 2. If the colors belonging to two adjacent units be mixed, an impression of both units is obtained which is plainly perceived as a mixture. 3. If two colors, not adjacent, be mixed, the intermediate color will tend to be brought before the mind, or white will be the result in the case of pure light, gray where there is partial absorption. 4. If any number of colors be mixed, the resulting impression will be that of a unit, a modified unit, or white. Dr. Edridge-Green exhibited and described a series of tests for color-blindness, which had been made for him, consisting of colored wools, silks, ribbons and cardboard.

Arterial Aneurysm Pressing on the Optic Commissure, Causing Distension of Optic Sheaths, Œdema of Retinæ, etc.—Mr. Jonathan Hutchinson, jr., read notes of the case of a man aged 29, affected with ulcerative endocarditis, who died in the London Hospital, under the care of Dr. Sutton. About twelve days before death he complained of rather sudden loss of sight in both eyes, vision being reduced to counting fingers. Upon examination, the retinal arteries were found extremely small, the veins much diminished, the retinæ white and hazy, and containing scattered hæmorrhages; these appearances remained unchanged till death. *Post mortem*, a small aneurysm was found, lifting up and pressing on the chiasma. It seemed to arise from the end of the basilar artery, and dipped into the pituitary fossa. Both optic nerve sheaths were greatly distended behind the globes, and the lymph spaces in the nerves and immediately beneath the pial sheath were much dilated. There was, in addition, slight retrobulbar neuritis; the central vessels were small but normal. Mr. Hutchinson considered that the case was of interest in connection with the view held by Deutschman and others, as to increased intracranial pressure and simple distension of the optic nerve sheaths not causing optic neuritis, but anæmia and œdema of the retinæ, with hæmorrhages. It seemed to agree with this theory, there being no intraocular neuritis, but only slight infiltration, with

leucocytes in the nerves behind the globes. During life it had been suggested that symetrical embolism of the retinal arteries was the cause of the amaurosis.

Dr. James Anderson was unable to agree with the conclusions of the writer, and did not think the case threw much light on the causes of optic neuritis. The aneurysm must have existed for some length of time, whereas the loss of sight came on suddenly, and shortly before death. Aneurysms of the cerebral arteries in association with rheumatism were not rare, but not necessarily accompanied by ocular symptoms. He thought the amaurosis in this case was probably a part of a general blood-infection such as occurred in cases of septic endocarditis.

Mr. Hartley mentioned a case in which double proptosis and uniocular optic neuritis had come on from three to six months after fracture of the base of the skull. The inflamed optic nerve passed into atrophy, and the proptosis slowly subsided. He had diagnosed an intracranial aneurysm pressing on the optic nerve.

Mr. Doyne looked upon the case Mr. Hartley had mentioned as one of retrobulbar neuritis, with consecutive changes in the discs. He did not think Mr. Hutchinson's case supported Deutschman's views, but was of opinion that the aneurysm had set up a localized meningitis, and that inflammation had extended from this down the optic nerves.

Dr. Herbert Habershon asked the cause of the aneurysm, whether due to arterial degeneration or embolism, and spoke of a case in which a cerebral artery became embolised, and an aneurysm formed on the cardiac side of the embolus.

Mr. Hutchinson, in reply, said the cerebral arteries had been carefully examined, and showed no further abnormality. He thought the fact that no abscesses formed, and no further changes occurred in the retinæ, went against the idea of septic infection. The symmetrical failure of sight was in favor of embolism.

Retinal Changes in Chronic Alcoholism.—Messrs. Edmunds and Lawford communicated the results of ophthalmoscopic

and microscopic examination of the retinæ of a man who died from alcoholic paralysis and heart disease. The ophthalmoscopic changes consisted of widespread haze of retinæ, without hæmorrhages or localized exudation. Sections of the retina revealed slight œdema of the nerve-fibre layer in the immediate vicinity of the optic disc, and well-marked œdema spaces in the outer granule layer, in which spaces were round and oval masses of clear homogeneous effusion. Attention was drawn to the rarity of this condition; the only recorded case, so far as the authors were aware, being one brought forward by Dr Sharkey, at the discussion on chronic alcoholism, at the Pathological Society, in December, 1888.

ERASMUS WILSON LECTURE ON THE PATHOLOGY OF GLAUCOMA.

BY PRIESTLEY SMITH, M.R.C.S.,

Ophthalmic Surgeon to the Queen's Hospital, Birmingham.

Delivered at the Royal College of Surgeons of England, March, 1889.

MR. PRESIDENT AND GENTLEMEN.—The healthy human eye presents to the educated touch of the surgeon a certain elastic resistance; its flexible walls are kept in a state of moderate tension by the fluids which traverse the chambers. Any eye which presents a decided variation from this characteristic condition, whether in the direction of hardness or of softness, is, as we know by experience, an unhealthy eye. I propose to speak in these lectures of the disorders which are associated with an increased tension of the eyeball, disorders to which we apply the name glaucoma.

With the advance of knowledge the word glaucoma has changed its meaning. In the early days of our art, it was applied without discrimination to the various diseases in which a gray or greenish blue appearance replaces the normal blackness of the pupil. In later times, when a clear distinction had been made between the opacities which lie in the crystalline lens and those which lie behind it, the name was reserved for some of the deeper seated and more destructive disorders, the nature of which remained unknown. Hardness of the eyeball, as a part of such disorders, received at that time only casual notice. In the year 1830, Mackenzie pointed out the frequent overfulness of the chambers in glaucoma, and made the first attempt to relieve that condition by puncturing the tunics. Twenty-five years later, von Graefe, studying the subject with

the aid of the ophthalmoscope, convinced himself that this overfulness was no mere complication of the disease, but was the essential cause of the leading symptoms, and was led by this conviction to his beneficent discovery of the curative action of iridectomy. Since that time the name glaucoma, losing its original meaning, has been used to indicate a disease characterized by increased tension of the globe. Certain other characteristics (cupping of the disc, contraction of the visual field) are recognized as consequences of the pressure.

Some writers, however, still deny that what we call glaucoma is necessarily connected with increased pressure. They point to certain exceptional cases in which the contracted field and the excavated disc are found in company with normal tension, and on the strength of these exceptions they maintain that glaucoma is the expression of some unknown agent which usually raises the pressure and excavates the disc at the same time, but which occasionally excavates the disc without raising the pressure. This assumption is, I think, unnecessary. The excess of pressure is sometimes slight; it is often intermittent; it may even be absent for long periods of time; it is very probable, therefore, that these cases of glaucoma with normal tension are cases which have been examined only during the intermissions of increased tensions. Permit me to mention a case in illustration.

Sir William Bowman was consulted by a lady in 1865. He noted (and he has kindly placed the notes at my disposal) increased tension and commencing excavation, diagnosed glaucoma, and spoke of iridectomy. No operation was performed. Twenty years later this lady came under my own care with deeply excavated discs, contracted fields and impaired vision. I saw her many times; sometimes the tension was quite normal, sometimes it was increased. Vision was still useful, but failing rather rapidly. After consultation with Sir William Bowman, iridectomy was performed on both eyes with good result.[1] Now this lady must have had for years an unmistak-

[1]Ophth. Rev. 1885, p. 261.

able glaucoma, but at times, and probably very frequently, a normal tension. Yet pressure was there at the beginning and it was there at the end. One cannot doubt that it was an essential factor in the process.

Glaucoma, then, may be broadly defined as *an excess of pressure within the eye plus the causes and the consequences of that excess.* To understand the morbid process in its entirety, we must study first the normal intra-ocular pressure and the processes by which it is maintained; secondly, the causes which lead to an excess of pressure; and, thirdly, the consequences of such excess.

Estimation of the Intra-ocular Pressure.—It will be well, in the first place, to consider the means by which the intra-ocular pressure has been measured or estimated.

Manometer.—In certain of the lower animals the pressure has been accurately determined by means of the manometer, an instrument which brings the intra-ocular fluid into direct communication with a column of mereury or other fluid. The average normal pressure, the variations due to pulse and respiration, and those produced by ligature of arteries and veins, by section and irritation of nerves, and by the action of certain drugs, have all been ascertained. The manometer has also been applied in a few cases to the living human eye, namely, to eyes doomed to excision, the measurement being made immediately before the operation.

Finger Test.—Under ordinary circumstances it is impossible to measure the pressure in the living human eye, and we have to content ourselves with measuring, as an index to it, the resistance of the tunics. We apply the tips of the two forefingers with light alternate pressure to the upper eyelid while the eye is directed downwards, and we record our observations by means of Bowman's symbols (Tn, T+1, T+2, etc.). For daily use no better method than the finger-test has been, or is likely to be, found, and it must be employed constantly by everyone who would deal successfully with diseases of the eye, but it is obviously inexact. We cannot state with precision the resistance which we feel, and we cannot rely upon the constancy of our sense of touch.

Tonometer.—To obviate this uncertainty, many attempts have been made to substitute for the fingers an instrument of precision—a mechanical tension-measurer or tonometer. I will not attempt to describe the various instruments which have been invented for this purpose; not one of them has hitherto found general acceptance. I will ask your attention rather to the principles which underlie and limit every effort of the kind, my object being not to show how every difficulty may be overcome, but to distinguish the possible from the impossible.

Principles of Tonometry.—Our object is to estimate the internal pressure from the behavior of the tunics under external pressure. The instrument, therefore, must press upon the eye, and must measure both the force employed and the impression produced, for it has more than one dimension; it varies in area and in depth. Are we to measure the area, or the depth, or both?

Area of Impression.—When a flat disc is loaded with a certain weight and placed upon a bladder, it flattens the membrane over a certain area; it sinks until it meets with precisely the same pressure from below as from above, then it remains in equilibrium; hence, if I employ a known external pressure, and measure the area of contact, I can calculate the internal pressure; for example, if I load a disc with a weight of 100 grammes, and if I find that the area of contact equals 100 square centimetres, I know that the internal pressure is 1 gramme to 1 square centimetre. If we were to take a second bladder of different size, or of different curvature at the point of contact, or of more extensible membrane, and place it at the same height and in connection with the same reservoir, we should find that the disc, if loaded with the same weight as before, would have the same area of contact as before. We see then that, *assuming that the membrane is perfectly flexible, the area of the impression produced by a known external pressure is a true index to the internal pressure.* It might appear, then, that the problem of ocular tonometry could be solved in this way. More than ten years ago I arrived by experiment at the principle which has just been stated, but concluded that the diffi-

culties of applying it to the eye were insuperable.[1] Quite recently Professor Fick of Würzburg, following the same theoretical considerations, has invented a tonometer which is correct in principle and very simple in construction.[2] A flat disc is pressed against the eye through the medium of a spring, and the force exerted by the spring is indicated by the scale behind it. The area of the disc is known; let us say that it is 50 square centimetres. I now press the disc against the bladder until it depresses and flattens exactly its own area of the membrane, neither more nor less, and I see by the scale that the force employed in doing this is, say, 50 grammes; I know that the internal pressure against the flattened area is equal to the external pressure, that is, 50 grammes, or 1 gramme to 1 square centimetre. Now the value of this instrument obviously depends upon one's ability to depress the membrane precisely to the extent of the disc. A small error in this respect gives a larger error in the estimate of the pressure; thus if the diameter of the depressed membrane be larger or smaller than that of the disc by one-tenth, the area will be at fault by one-fifth, for the areas of circles vary as the squares of their diameters; and the estimate of the internal pressure will be at fault to the same extent. In the actual instrument the diameter of the disc is about 7 millimetres, so that the difficulty of applying it with precision to the eye, covered as the latter is by a more or less compressible conjunctiva, is considerable. Moreover, the readings of the instrument are at the mercy of the operator's wish or prejudice, and it is difficult to apply it with strict impartiality when a little more or a little less pressure will confirm or falsify one's own opinion. The principle of estimating the internal pressure from the area of the impression is unquestionably the right one, and Professor Fick's tonometer is the only one clearly based on this principle. I fear, however, that the difficulty of applying it with precision will be found to cause uncertainty in the results obtained.

[1]Glaucoma, its Causes, etc. London, Churchill, 1879, p. 48.

[2]Inaugural Dissertation by R. A. Fick, Würzburg, 1888, and Transactions of International Ophthalmic Congress, Heidelberg, 1888, p. 289.

Depth of Impression.—The depth is more easily measured than the area. But the sclera of the human eye varies somewhat both as to curvature and extensibility in different individuals, hence *the depth of the impression produced by a known external pressure is not a perfectly true index to the internal pressure.*

And there are other sources of error which cannot be avoided, whichever method we employ. The tunics of the eye are not perfectly flexible; their rigidity varies at different parts and in different inviduals. Again, the thickness of the conjunctiva must be taken into account; a swollen conjunctiva pits deeply, irrespective of the yielding of the sclera. And again, it must be remembered that the tonometer itself raises the intra-ocular pressure for the moment, so that even if it could measure the internal pressure accurately we should learn, not the pre-existing pressure, but the pressure as modified by the application of the instrument; the amount of this modification remains unknown, and it varies in different cases according to the ease with which fluid is extruded from the eye.

No tonometer, then, however ingeniously devised, can accurately measure the intra-ocular pressure, or accurately compare one eye with another. (Here follows the description of the author's own tonometer).

The manometer, the finger test, and the tonometer in combination, have been useful in determining the pressure which is normally present in the living human eye. If we connect the eye of the dead subject by means of a hollow needle and elastic tube with a column of mercury or other fluid, we can ascertain what pressure is required to induce a degree of tension resembling that of the living eye; we can compare the artificial with the natural tension, either with the finger or more accurately with the tonometer. In like manner we can ascertain what pressures appear to correspond with our own ideas of T+1, T+2, T+3, or with the various degrees upon the scale of the tonometer.

Amount of the Intra-ocular Pressure.—The foregoing are the means by which the intra-ocular pressure has been estimated.

We may next consider the results obtained. Concerning the normal intra-ocular pressure in the eyes of animals, we have a considerable mass of evidence and a very close agreement. The animals chiefly experimented on were rabbits,cats and dogs, and the precautions taken against error were very minute. In nearly all cases the pressure was between 20 and 30 millimetres of Hg; the average was about 25 millimetres. Slight oscillations were found to accompany pulse and respiration, and a more marked rise was produced by contraction of the external muscles of the eye.[1]

Experiments upon the dead human subject with my own tonometer gave a closely corresponding result. I found that in order to produce a tension resembling that of the living eye, a pressure of about 30 centimetres of water—that is, 25 millimetres of Hg— must be employed, and the same result was obtained on testing with the finger. Other observers employing the tonometer in the same way have placed the normal pressure rather higher than this, in one case even as high as 40 millimetres of mercury; but that this latter figure presents a distinctly glaucomatous tension will be manifest to anyone who will take the trouble to employ the finger test in the mortuary in the manner described. Adolph Weber, trusting the finger rather than the tonometer, estimates the pressure, as I have done, at about 25 millimetres of Hg.[2]

This estimate has lately been confirmed by Wahfors, who applied the manometer to several human eyes immediately before excision. One of these was a healthy eye, which had to be sacrificed on account of an oribital tumor. The intra-ocular pressure was equal to 26 millimeteres of mercury.[3]

Again, in the lower animals, numerous experiments with the double manometer have proved that the pressures in the aqueous and vitreous chambers are equal, or so nearly equal that the difference is not discoverable.[4] It has been asserted that

[1]Graefe-Saemisch, Handbook, vol. 2, p. 371.
[2]Glaucoma, its Causes, etc., p. 98.
[3]Trans. of Internat. Ophth. Congress, 1888, p. 268.
[4]Bellarminoff, Abstract in Ophth. Rev., 1887, p. 361.

the vitreous pressure is considerably higher than the aqueous pressure, and some observers have even declared themselves able to detect the difference by placing the fingers on different parts of the globe. This idea, though disproved by accurate observation, is not yet quite abandoned. Experiments of my own upon the freshly excised eyes of animals have shown that the lens is displaced from its normal position by a slight excess of pressure either before or behind it. Thus, an excess in the vitreous chamber equal to 5 millimetres of Hg causes a considerable displacement of the lens and iris, and an excess of 10 millimetres almost abolishes the anterior chamber.[1] Such differences in the two chambers are quite too small to be detected by the finger, and it is obvious that any comparison of the scleral with the corneal tension by means of the tonometer is fallacious. When such differences do occur in the living eye, they produce, and are manifested by, displacements of the lens and iris forward or backward as the case may be.

Concerning the height to which the intra-ocular pressure rises in the human eye under abnormal conditions we have very little evidence. In a case of chronic glaucoma, in which the tension had been slightly reduced by an unsuccessful iridectomy, Wahlfors found with the manometer a pressure of 71 millimetres of mercury. Experiments with my own tonometer seem to indicate that the tension which we call +3 corresponds to a pressure considerably higher than this. Much higher pressures have been produced experimentally in the eyes of animals. In one instance a pressure equal to 200 millimetres of mercury was produced by compression of the aorta and simultaneous irritation of the nerve.[2]

Secretion and Excretion of the Intra-ocular Fluids.—The maintenance of the normal pressure in the chambers of the eye depends upon the due secretion and the due excretion of

[1]Ophth. Rev., 1888, p. 207.

[2]Von Hippel and Gruenhagen. Von Graefe's Archiv., vol. xiv, part 2, p. 219. See also Glaucoma, its Causes, etc, p. 102.

the fluids which traverse them. These physiological processes have been most carefully studied during the past fifteen years, and it will be well to review the evidence upon which our knowledge stands at present. It is only by a careful study of these processes that we can hope to unravel the complex pathology of glaucoma.

The intra-ocular fluids flow, like all other secretions, from the blood-stream. Studying their origin from the anatomical point of view, we find, as possible sources, two vascular membranes, the uveal track and the retina. We may at once eliminate the retina, for clinical observation shows that complete blockage of the retinal vessels by embolism causes no discoverable change in the fulness of the chambers. The uveal tract consists of three distinct parts, namely, the choroid extending forward as far as the ora serrata, the ciliary portion reaching from the ora serrata to the base of the iris, the iris ending at the margin of the pupil. The functions of these several parts may to some extent be inferred from their structure and relations.

The choroid, by its internal surface, nourishes the external layers of the retina. Its capillaries feed the pigmented epithelium, and the epithelial cells keep the rods and cones in a state of functional activity. The meshes of the capillary plexus become progressively less and less close from the posterior pole to the anterior limit, and this arrangement accords with the varying sensibility of the corresponding zones of the retina. There is no evidence that the choroid nourishes the vitreous body, and it is obviously improbable that a highly organized membrane like the retina, which has many differentiated layers and a separate vascular system of its own, should convey nourishment from the choriod to the vitreous.

The iris has a well-defined optical function, that, namely, of regulating the entrance of light. It is possible that its posterior surface, like the rest of the uveal surface, may have a secretory function as well, and take some part in the formation of the aqueous humor, but this part must be quite a subordinate one, for there are many cases on record in which the iris has

been completely absent, either from birth or from injury, and there has been no discoverable insufficiency of the aqueous fluid; the tension has been normal.

The ciliary portion of the uveal tract shows a special adaptation for the supply of fluid to the vitreous body, the lens, and the aqueous chamber. Where it is in relation with the vitreous, its secreting surface is extended by a series of grooves and ridges, and where it is in relation with the aqueous chamber it assumes a still more convoluted arrangement well suited for rapid secretion.[1]

The vitreous body has its chief attachments in this region; its limiting membrane adheres firmly to the ora serrata, and in advance of this point is separated from the secreting surface only by a single layer of cylindrical cells; the membranous septa which spring from the limiting membrane in this region are arranged in a manner peculiarly favorable for the entrance of fluid.[2]

Pathological anatomy points to the same conclusion. If we examine eyes which have been excised during the first stage of vitreous infiltration, we find an inflammatory exudation entering the vitreous body from this portion of the uveal tract, the region of its influx being limited posteriorly by the ora serrata. We find also that while a shrinking vitreous readily separates from the retina it always retains its firm connections in the region of the ora serrata. And, again, we find that while very extensive atrophy of the choroid does not necessarily affect the transparency or the volume of the vitreous body, disease in the ciliary region always tends to its destruction.

Experiments upon animals have amply confirmed the inferences previously drawn from these anatomical and pathological facts. Deutschmann found that removal of the ciliary processes together with the iris, which can be done in the rabbit without loss of lens or vitreous, and without causing inflammatory destruction of the eye, is followed by a total arrest of the secretion of the aqueous, and by atrophy of the vitreous

[1]See description by Brailey, Journal, September, 1882, p. 577.
[2]See description by Straub, Von Graefe's Archiv., vol. 24, part 4, p. 7.

body and lens.[1] Schoeler, Uhthoff, and others have found that after subcutaneous injections of fluorescine there is a speedy coloration of the aqueous fluid, and a more gradual coloration of the vitreous body, and that the colored secretion proceeds from the ciliary processes, and perhaps to a small extent from the posterior surface of the iris also.[2] Leplat has demonstrated the same thing by a somewhat different method; he gave subcutaneous injections of iodide of potassium, enucleated the eyes after various intervals of time, froze them and divided them into zones, and made a quantitative test for iodide in each zone.[3] These and many other experiments agree in showing that the fluids which nourish the vitreous body and lens and fill the aqueous chamber are secreted chiefly, if not entirely, by the ciliary portion of the uveal tract.

In what direction do these fluids travel, and where do they escape from the eye? The aqueous fluid passes from the posterior chamber forward through the pupil into the anterior chamber. It has been asserted that there is a current passing forward through the base of the iris, but the evidence is by no means conclusive. It is contrary to physical reasoning to suppose that while the pupil remains open the fluid will traverse the tissues of the iris: moreover, the stream through the pupil is proved by the fact that when the pupillary margin becomes entirely adherent to the capsule the fluid collects behind the iris with disastrous consequences.

The aqueous fluid escapes from the eye at the angle of the anterior chamber, by filtering through the ligamentum pectinatum into Schlemm's canal and the veins connected with it.

This fact was established by Leber's well-known injection experiments, which showed also that the anterior chamber has no direct communication with the blood-vessels, and that the cornea, so long as its posterior epithelium remains entire, is permeated by the aqueous fluid.

The escape of fluid from the vitreous body has been the sub-

[1]Von Graefe's Archiv., vol, 26, part 3, p. 117, and Ophth. Rev., 1882, p. 149.
[2]See Ophth. Rev., vol. i p. 413.
[3]See Ophth. Rev., 1888, p. 84.

ject of much experiment and discussion, and is still not determined with absolute certainty. The question is whether it escapes at the papilla, or through the aqueous chamber, or in both these directions. Schwalbe has described certain lymph passages within the sheath of the optic nerve, which, he says, "find exit in the lymph passages of the skull, and convey not only the lymph formed in the optic nerve, but that also of the retina and vitreous."[1] Stilling has seen a considerable escape of fluid in this region under artificial pressure.[2] The injection experiments of Leplat, already referred to, appear to show that iodide of potassium injected subcutaneously enters the vitreous from the ciliary body and travels very slowly backward, and leaves it at the papilla.

Still more positive evidence is afforded by some experiments made by Gifford.[3] He injected with special precautions small quantities of water containing Indian ink or cinnabar in suspension into the vitreous of rabbits and other animals. The results were definite and constant. On the second day, or later, according as the injection was made farther backward or forward in the vitreous, the ophthalmoscope showed the particles collecting in the excavation of the papilla. On killing the animal a day or two later, the microscope showed the particles passing backward through the papilla, along the lymph spaces around the central vessels and leaving the nerve trunk with the vessels, and passing toward the sphenoidal fissure. They did not enter the sheath of the optic nerve. The current within this sheath has been proved by other experiments to move from the brain toward the eye.

In spite of the evidence put forward by these investigators, it has been difficult to accept the view they advocate, for certain facts seem to point the other way. Schoeler found that artificial occlusion of the supposed posterior outlet caused no discoverable reduction in the amount of fluid escaping from

[1]Graefe-Saemisch, Handbook, vol. i, p. 50.

[2]Report of Heidelberg Congress of 1885, p, 42.

[3]Archives of Ophthalmology, 1886, p. 153, and Ophth. Rev., 1886, p. 217.

the eye in a given time.[1] More recently, in experiments made by himself and Uhthoff, he failed to find any transit of fluorescine from the eyeball into the optic nerve or its sheaths. I myself, ten years ago,[2] injected solutions of carmine and of aniline purple into the vitreous of freshly excised pig's eyes, maintained the pressure for five or six hours, then froze the eye, and cut microscopic sections through the posterior pole; there was no color deeper than the superficial layers of the retina. Again I injected a colored fluid into the sheath of the optic nerve, after ligaturing the nerve behind the point of injection. The pressure employed was about double the normal intra-ocular pressure, and was maintained at this height during five hours. Microscopic sections showed no trace of fluid having entered the eyeball, although the tissues were deeply stained up to the anterior extremity of the sub-vaginal space.

In favor of the supposition that the vitreous fluid escapes through the aqueous chamber is the fact that it can easily pass in this direction. Leplat has lately proved, with regard to the living eye, that the fluid which rapidly refills the aqueous chamber after it has been emptied by puncture of the cornea is derived in large measure from the vitreous chamber; and this same refilling of the aqueous chamber occurs also in the dead eye, in which the vitreous is the only possible source of the fluid.

In order to test this matter still further, and to obtain, if possible, some information as to the amount of fluid which traverses the chambers in a given time, I undertook last year[3] another series of injection experiments on the freshly excised eyes of oxen, sheep and pigs, and, in a few instances, on the human eye after death. The most trustworthy results were obtained with sheep's eyes, for I could have an unlimited supply of these within a few minutes of death. The apparatus employed enables one to make a continuous injection of one

[1]Von Graefe's Archiv., vol. 25, part 4.
[2]Glaucoma, its Causes, etc., p. 142.
[3]Ophth. Rev., July, 1888, p. 193.

or both chambers under a known and constant pressure, and to measure the amount of fluid which passes through the chambers in a given time. The pressure can be varied at will by raising or lowering the reservoirs, and it can be kept absolutely equal in the two reservoirs by opening the tube which connects them; or, when this tube is closed, the reservoirs can be adjusted to give different pressures.

An air bubble in each of two graduated horizontal tubes serves to indicate the movement of the fluid. Its position is noted at regular intervals of time. The cubical content of the tube is known. A pressure of 30 centimetres of water, that is, 25 millimetres of mercury, was adopted as the standard pressure throughout, this being about the normal pressure.

The experiments were of several kinds. In one series I injected the anterior chamber only; in another the vitreous chamber only; in a third series, aqueous and vitreous were injected simultaneously under precisely equal pressures; and in a number of other cases I employed different pressures in the two chambers simultaneously, and varied the conditions in many different ways. The details have been described elsewhere; here I need only deal with the results. The following were the averages of ten experiments of each kind; the variations were not great:

When the anterior chamber only was injected, the escape during the first half hour was 785 cubic millimetres.

When the vitreous only was injected it was 275 cubic millimetres.

When the aqueous and vitreous were injected simultaneously under the same pressure it was 485 cubic millimetres, that is, considerably less than during injection into the aqueous chamber only.

This result, at first sight paradoxical, is explained by the varying position of the iris, and its effect upon the patency of the angle of the anterior chamber—the "filtration angle." When the anterior chamber only was injected, the iris was displaced a little backward; when the aqueous and vitreous

chambers were injected simultaneously under equal pressures, the iris remained in its normal position, or nearly so; when the vitreous only was injected the iris was displaced a little forward. In order to observe these displacements accurately, I froze the eyeballs solid while the injection was still going on, and bisected them.

In the fourth series of experiments the vitreous reservoir was raised to a higher level than the aqueous reservoir. For example, keeping the aqueous reservoir at 30 centimetres, as before, the vitreous pressure was raised to 35 centimetres. Under these circumstances the diaphragm between the two chambers, consisting of the lens, zonula, processes and iris, is displaced forward until the increased tension of the zonula balances the excess of pressure behind it. Equilibrium being established in the diaphragm, a stream passes through the zonula from the vitreous to the aqueous chamber, and fluid continues to enter the eye through the one needle, and to leave it through the other. The amount which escapes by filtration is then ascertained by subtracting the backward movement of the one bubble from the forward movement of the other. Now, under these circumstances, although the pressure in the aqueous chamber was as high as in the previous experiments, and the vitreous pressure was actually higher, the total escape was reduced to about 50 cubic millimetres in the first half hour. In the frozen and bisected eye the iris-base was found to be pushed forward, and in places visibly in contact with the periphery of the cornea. By placing the vitreous reservoir 15 centimetres higher than the aqueous reservoir it was possible to empty the anterior chamber almost completely, and to apply the whole iris close to the cornea; and under these circumstances the escape by filtration from the whole eye appeared in some instances to be entirely arrested.

Similar experiments upon the freshly-excised eyes of oxen, and in one instance on a freshly-excised healthy human eye removed with an orbital tumor, gave closely similar results.

With regard to the escape at the papilla I made some

further experiments with the smaller apparatus. A circular piece, including choroid, sclera, retina and optic nerve is cut from the back of a sheep's eye immediately after death and placed in the box, and the inner tube is tightly pressed down upon it by screwing on the cap of the box. The inner tube is then filled with colored fluid and connected by a flexible tube with a graduated glass tube and reservoir, as in the other apparatus. Under a pressure of 30 centimetres, as before, there was no perceptible exudation and no coloration on the outer surface after several hours.

These experiments show several points of importance with regard to the freshly excised eye. They show, firstly, that fluid injected into the vitreous chamber escapes chiefly, if not entirely, through the aqueous chamber; secondly, that a slight excess of pressure in the vitreous chamber displaces the lens and iris forward, compresses the filtration angle, and impedes the escape of fluid; thirdly, that there is little, if any, escape at the papilla. With regard to the currents in the living eye, they give, of course, no positive information.

(Since this lecture was delivered I have read with much interest that Dr. Leplat, adopting a method nearly the same as that above detailed, has made a further series of experiments upon living animals. His results confirm to a large extent those obtained by myself with the freshly-excised eye. He concludes that the escape at the papilla is about one-fiftieth of the escape at the filtration angle.)[1]

If we compare these various observations concerning the movements of the vitreous fluid, it is not difficult, I think, to reconcile the apparent discrepancies.

It appears certain, from the movements of solid particles observed by Gifford, that there is a current passing backward in the vitreous body and escaping along the lymph-passages which surround the central vessels of the optic nerve. It appears equally certain that this current moves very slowly, and that the amount escaping in a given time is extremely small

[1]Annales d'Oculistique, January February, 1889.

as compared with that from the aqueous chamber. Whether the small perivascular spaces in the papilla are rendered impervious by excision of the eye, or whether a small quantity of fluid does actually traverse them during artificial injection of the vitreous chamber, it is difficult to determine. In any case the absence of visible filtration here presents a striking contrast with the free escape which occurs at the filtration angle. It remains uncertain, I think, whether there is a persistent stream from the vitreous to the aqueous chamber. But we have seen that fluid can pass very readily in this direction under a slight excess of pressure in the vitreous chamber; and it seems probable that so long as the partition remains permeable, and the fluid diffusible, any excess of pressure in the vitreous chamber will relieve itself in this way. An intercommunication of this kind between the two chambers appears to be necessary for the maintenance of the lens in its normal position.

Our knowledge of these currents may, then, be briefly summarized as follows: The fluids which nourish the vitreous body and lens, and fill the aqueous chamber, are secreted chiefly by the ciliary portion of the uveal tract. The larger part of the secretion passes directly into the aqueous chamber, forward through the pupil, and out at the filtration angle. A very much smaller portion passes backward through the vitreous body, and escapes at the papilla. The hyaloid membrane and zonula which separate the two chambers are readily permeable by the vitreous fluid. The pressure which the fluid exerts against the walls of the chambers is equal to about 25 millimetres of mercury, and is the same or nearly so, in the two chambers.

To conclude this physiological part of the subject, I may point out that while the circulation of the intra-ocular fluid is necessary to the eye as a living organ, its pressure is necessary to it as an optical instrument—an instrument which is built entirely of soft materials, but which, nevertheless, can adjust itself with precision both for direction and for distance. In the larger chamber, where there are no moving parts,

the fluid is supplemented by a transparent fibrous tissue, which adds to the safety of the organ. In the smaller chamber there is fluid only, so that the curtain which regulates the admission of light may have perfect freedom of movement. In both chambers the fluid exerts that particular degree of pressure which suffices to maintain the form of the globe, and gives precision to the action of the muscles, external and internal, but does not embarrass the circulation of the blood, and nutrition of the tissues, or the transmission of nerve currents within the organ.

When the intra-ocular pressure rises above these physiological limitations, we get that complex disturbance which we call glaucoma.

Predisposing Causes of Primary Glaucoma.—The ciliary processes are highly vascular and vary in size, according to the quantity of blood in their vessels. If the free space at their disposal be insufficient for these variations, they will at times press unduly against the adjacent parts, internally against the lens margin, anteriorly against the iris-base. I think it can be shown that an undue proximity between the margin of the lens and the surrounding parts, in other words, an insufficient circumlental space, predisposes the eye to glaucoma. This idea was propounded in a work of my own published ten years ago.[1] It was put forward at that time as a hypothesis, and it has served the true purpose of every hypothesis, good or bad —it has suggested fresh lines of inquiry.

Supposing the circumlental space in any given eye to be insufficient, the fault may lie presumably either in the lens, or the parts which surround it. We know that the liability to glaucoma is greater in the old than in the young; the question therefore arises: Does the relation of the lens to the surrounding parts alter with the advance of life? In the year 1880 I examined, with regard to this point, five pairs of healthy eyes taken from adult male subjects, varying in age from 21 to 90, and I found a well-marked increase in the size of the lenses

[1]Glaucoma, its Causes, etc., London, 1879.

from the youngest to the oldest.[1] A more thorough investigation naturally followed; 156 lenses removed from the dead subjects were examined. They belonged, in nearly equal numbers, to the six decades of life between 20 and 80, and in smaller number to the decade 80 to 90. Each lens was accurately weighed, and then measured as to its volume, by means of an apparatus devised for the purpose. In most cases the linear dimensions were measured also. Opacity when present was noted. The specific gravity was calculated in each case from the weight and volume. Details of the method and of the precautions taken against error have been described elsewhere.[2] Here it is only necessary to state the results.

The crystalline lens, so long as it remains healthy, increases in weight and in volume throughout the whole of life. During the forty years between 25 and 65 years of age, it adds about one-third to its weight, one-third to its volume, and one-tenth to its diameters. The specific gravity appears to vary a little in individual cases, but shows no decided change with the advance of life. Lenses which are becoming cataractous are, as a rule, smaller than healthy lenses belonging to the same period of life. These are anatomical facts; physiology explains them.

The lens is derived from the cuticular epiblast, and in its mode of growth is analogous to the cuticle. But its cells, unlike those of the cuticle, are not cast off as they grow old; they are laid down layer upon layer within a closed capsule, the younger fibres surrounding the older. In consequence of this unique arrangement, and in spite of the shrinking of the older cells, which form the nucleus, the growth of the lens does not cease with that of the rest of the body, but is continuous, unless some morbid process intervene, thoughout the whole period of life. In advanced life the process of growth often fails; then the shrinking nucleus tends to separate from the softer cortex,[3] and senile cataract begins; accordingly, the lens

[1] R. Lond. Ophth. Hosp. Reports, vol. x, p. 33.
[2] Trans. Ophth. Soc., 1883.
[3] Becker, Anatomy of the Healthy and Morbid Lens, see Ophth. Rev., vol. ii.

with incipient cataract is smaller than the healthy lens of the same age.

Now, the structures which surround the lens attain their full dimensions at the commencement of adult life, or even earlier. I shall have to point out immediately that the diameter of the cornea increases little, if at all, after the fifth year. Hence it comes about that as age advances the lens steadily encroaches upon the space in which it lies. Its margin comes into closer relation with the ciliary processes. The precise relation of these parts in the living eye cannot be ascertained, for it is altered by death and by excision, but the change in question is obvious when we examine eyes of very different ages. Its anterior surface approaches nearer to the cornea, and thereby diminishes the depth of the anterior chamber. The shallow anterior chamber of old age has been supposed to indicate an advance of the whole lens toward the cornea, but such an advance has not been proved or explained, and the idea is not reconcilable with the change of refraction which actually occurs. A simple advance of the lens in an emmetropic eye would cause myopia, whereas the tendency in advanced life is to hypermetropia.

The increased size of the lens is itself, I think, to a large extent, the cause of the acquired hypermetropia of advanced life. If a lens be enlarged symmetrically—that is, if its several diameters be increased in equal proportion—its focal length will be increased in the same proportion. Other things being equal, therefore, the focal length of the human lens must increase as its size increases. The result may, of course, be modified by changes in the index of refraction, or by disproportionate enlargement in one or other diameter; but it is clear that the growth of the lens cannot be omitted, as hitherto, from the calculation. The dimensions of an aged but healthy lens are very different from those which are given in books as the average dimensions of the adult lens; thus, in the Graefe-Saemisch *Handbook* (vol. i, p. 45) the average antero-posterior diameter is given as 3.7 millimetres, whereas in elderly persons I have found it 6, 6.5, and even 6.75 millimetres.

As the result of this inquiry the glaucoma question stood thus: The lens steadily increases in size as life advances. If the size of the lens is an important factor in glaucoma, the liability to glaucoma should steadily increase in like manner. Does it so increase? With the help of many friends, members of the Ophthalmological Society, I was able to collect accurate data concerning 1,000 cases of primary glaucoma.[1] The cases were tabulated on a uniform system, indicating the sex of the patient, the age at which the glaucoma began, and the type of the disease, whether chronic, subacute or acute. The figures so obtained indicated the relative frequency of glaucoma in the several decades of life. These figures were then adjusted to the number of persons, males and females, living in each period of life, and thus the liability belonging to each sex and each life-period was ascertained. The following are the salient points.

A. *Frequency.*—1. Primary glaucoma is extremely rare in childhood and youth. Not one per cent of the cases met with begins earlier than the 20th year (5 per 1,000 belong to the second decade). 2. Its frequency increases, slowly at first, more rapidly later on, in each decade, until about the 60th year; between 60 and 70 it is about as frequent as between 50 and 60; after 70 its frequency diminishes. 3. Cases beginning after 50 are about twice as numerous as cases beginning before 50 (679 and 321 per 1,000.) 4. Females suffer in rather larger numbers than males (569 and 431 per 1,000). 5. The chronic non-congestive form is rather commoner in males than in females (253 and 223 per 1,000). 6. The acute and subacute congestive forms are much commoner in females than in males (346 anb 178 per 1,000).

B. *Liability.*—7. The liability to primary glaucoma is extremely slight in childhood and youth as compared with the later periods of life; thus at 15 years of age it is at least a hundred times smaller than at 65. 8. It continually increases up to and during the seventh decade, that is, the ten years be-

[1]Trans. Ophth. Soc., 1886.

tween 60 and 70; between 60 and 70 it is more than twice as great as between 40 and 50. 9. After 70 years of age the liability to glaucoma appears to decline considerably; it is probable, however, that the statistics relating to this period do not correctly represent the frequency of the disease, for very old people do not seek advice so easily or so willingly as those who are younger. 10. The liability of females is greater than that of males in a ratio probably of about 6 to 5. 11. The extra liability of females pertains to the whole of life, except, perhaps, the periods before 30 and after 70, concerning which the data are too few to justify a generalization. 12. The extra liability of females relates very markedly to the congestive forms of the disease, not to the non-congestive.

There is, then, a certain parallelism between the increasing liability to glaucoma and the increasing size of the lens, which accords well with the supposition that a diminished circumlental space predisposes to glaucoma.

Permit me now to ask your attention to another anatomical fact which seems to point in the same direction. Every operator must sometimes have been struck, when performing an iridectomy for glaucoma, by the apparent smallness of the cornea. The idea suggested itself that a small cornea might indicate a small globe, or at least a small ciliary zone, and might thus point to another cause of insufficiency in the circumlental space. I have endeavored to elucidate this point by systematic measurements of the cornea in healthy and in glaucomatous eyes. The instrument employed is a keratometer devised for the purpose. It combines a millimetre scale with a convex lens, and, by a simple optical principle, enables one to measure accurately without touching the eye or alarming the patient. I have not attempted to measure to less than half a millimetre.

Fifty-four persons suffering in one eye or in both from primary glaucoma, and 330 persons whose eyes were healthy, apart from trivial affections and errors of refraction, were examined. The measurements were tabulated according to sex, age and refraction. The list contains many cases of high hypermetro-

pia and high myopia. The results may be generalized as follows:

The cornea of the healthy eye, or at least its visible part, attains its full diameter very early in life. Between 5 and 10 years of age the average horizontal diameter is 11.6 millimetres; between 10 and 20, and again between 20 and 40, it is exactly the same. After 40 it appears to be slightly smaller, namely, 11.4 millimetres. The dimension varies in individual cases; but at all ages from 5 to 90 it usually lies between 11 and 12 millimetres. (In four persons out of the 330 it was 12.5, and in one 13.5. In eight persons it was 10.5. These eyes appeared perfectly healthy, and were the only exceptions). Small corneas are met with, of course, together with congenital cataract and nystagmus in obviously microphthalmic eyes, but such cases are not included in my list.

The average diameter is the same in the two sexes, and it is the same in hypermetropic, emmetropic, and myopic eyes. There is no difference in the average, even between highly hyperopic and highly myopic eyes. There is very rarely any difference between the two eyes of the same individual, and this is true even when they differ widely in refraction.

In persons suffering from primary glaucoma the cornea is often small, that is, the horizontal diameter measures 10.5 millimetres or less. Among the glaucoma patients examined it was small in 32 per cent. Among other persons of more than 40 years of age it was in only 5 per cent. To put the matter in a more striking way; 28 persons out of the whole number examined had small corneas, and of these 28, 20 had glaucoma; moreover, in 6 of these the cornea measured only 10 millimetres, a smaller dimension than was found in any unaffected person.

Those who had small corneas at all had it in both eyes, though only one eye might be glaucomatous. Thus, a man (J. D.), aged 66, had a chronic non-congestive glaucoma of many months' duration in the left eye; the right was perfectly healthy. Both eyes were emmetropic; both corneas measured 10 millimetres horizontally. Such cases prove that the

smalless of the cornea precedes the glaucoma and is not caused by it.

In four cases both corneas were small, but one was smaller than the other; in each case the smaller cornea belonged to the glaucomatous or to the more glaucomatous eye.

The small cornea is often oval in shape, the vertical diameter being shorter than the horizontal. I am not sure whether this oval shape is more frequent among small corneas than among those of full size.

Since my attention was first directed to the point it has happened to me several times to be struck, on first glancing at a patient, by the smallness of the cornea, to suspect the presence of glaucoma on that account, and to find it in one or both eyes. As to the frequent association of the two conditions there is no doubt; as to the meaning of it I cannot speak with certainty.

Are these small corneas small from youth upward, or do they become so in later life? We know that the cornea is nourished by the vessels of the conjunctiva and sclera, and that in shrinks to a very small size in some destructive diseases of the eye; its seems possible, therefore, that even in good eyes it may shrink a little when senile changes in other tissue sets in. As a fact, small corneas seem to be commoner in elderly than in young people. On the other hand, they are not entirely absent even among the young, and may in the young also be associated with glaucoma. At a meeting of the Ophthalmological Society on March 11, 1886, Mr. Hartridge showed a case of non-congestive primary glaucoma in a girl aged 14—a very rare condition at that time of life. One eye only was affected. Both corneas were strikingly small. Mr. Hartridge kindly measured them afterward, at my request. The diameters were 10 millimetres vertically, 10.5 millimetres horizontally.

The term "microphthalmos" denotes that the eyes are very obviously below the normal size, and in such eyes the small cornea is often associated with a shrunken and degenerated lens, but not always. Microphthalmic eyes with healthy

lenses are, I believe, very prone to become glaucomatous, and in one such eye examined by Hocquard and Masson,[1] the lens was found to be much too large for the eye; speaking more correctly, the eye was much to small for the lens.

In some of my own specimens I believe that a similar disproportion between the lens and its surroundings may be made out, though the point is difficult to determine. Further, I may remind you that in one form of secondary glaucoma an alteration in the size of the lens certainly plays an important part; the swelling which follows laceration of the lens capsule, either by accident or by operation, is very apt to induce glaucoma, and more so in elderly people, in whom the lens is already large, than in children in whom it is comparatively small.

Again, there is a third anatomical condition which, when present, must diminish the circumlental space—excessive size or prominence of the ciliary processes. Unfortunately, we cannot see these organs in the living eye, and we cannot learn their antecedent condition from eyes which have been blinded by glaucoma. We can, however, gain important evidence from eyes which have suffered from glaucoma, and have been quickly cured. Such evidence is to be found in a case described by Fuchs.[2] The patient was a woman aged 66; both eyes were attacked by acute glaucoma at intervals of one year, and both were permanently cured by iridectomy. They were examined after death several years later. In both eyes the attack had been of short duration; in the second eye only two days, so that, except for the coloboma, it had probably altered the conditions of the eye but little. I extract the following points from the published description: The ciliary body showed a remarkable enlargement, both as regards the muscle and the processes. The processes extended almost to the lens on the one side and to the iris on the other. The angle of the anterior chamber was considerably narrowed by the altered position of the iris-base, so that a slight swelling of the

[1] Archives d' Ophth., 1883, p. 231.

[2] Von Graefe's Archiv. vol xxx, part 3, p. 123

processes would have pressed the iris against the cornea. The circumlental space was remarkably small, not through of the ciliary processes. The tissues of the ciliary body were normal, not inflamed. The condition was simply a hyperplasia, or perhaps only a physiological peculiarity.

In this connection it must be noted, also, that the hypermetropic eye, the ciliary muscle of which is especially prominent in the direction of the lens, is particularly liable to glaucoma. These facts appear to justify the belief that an insufficient circumlental space predisposes the eye to glaucoma. The insufficiency may depend upon several causes acting singly or in combination. First, there is the increasing size of the lens, which explains the increasing liability which comes with advancing years. Secondly, there is, in some cases, the subnormal size of the globe or of the ciliary zone, sometimes depending on insufficient growth, sometimes possibly on senile contraction. Thirdly, there is the excessive size or prominence of the ciliary processes, a condition which may perhaps be present at any time of life, but which will certainly be more likely to produce complications in advanced life, when the lens is large, than in youth, when it is small.

There are doubtless other prodisposing causes, but we have at present, I think, no certain knowledge with regard to them.

Exciting Causes of Primary Glaucoma.—For these we must look to conditions which overfill the uveal tract with blood. The common antecedents of glaucomatous attacks are conditions such as heart weakness, bronchitis, hepatic congestion, constipation, cold, hunger, bodily fatigue, mental exhaustion and others, which congest the venous system. Again, the arterial hyperæmia which accompanies some forms of trigeminal neuralgia, or which follows a scratch or contusion of the eye itself, will suffice to light up a glaucoma where there is a strong predisposition to it.

Congestion of the uveal tract involves enlargement of the ciliary processes, and this, if it be extreme, or if the space at the disposal of the processes be insufficient, leads to compres-

sion of the infiltration angle. An advancement of the lens toward the cornea usually accompanies the onset of the high tension; the cause of this is not quite clear. It is probable, I think, that the ciliary processes being compressed between the lens and iris secrete an undue proportion of fluid into the vitreous chamber, and that the filtration of fluid from the vitrous to the aqueous chamber is impeded. In some forms of glaucoma, especially the chronic form and that which sometimes accompanies the advanced stage of retinitis pigmentosa, there are, I suspect, changes in the hyaloid and anterior part of the vitreous which obstruct the escape of surplus vitreous fluid into the aqueous chamber, and tend thereby to enlarge the vitreous at the expense of the aqueous chamber. Hæmorrhagic and serous exudation into the vitreous chamber appear sometimes to act in the same way. In any case the advance of the lens and zonula is an important factor. The experiments described in my first lecture showed that a very slight excess of pressure in the vitreous chamber drives the lens and ciliary processes forward, compresses the filtration angle, and checks the escape of the fluid from the eye.

Acute glaucoma is often spoken of as an inflammatory disease, and undoubtedly its course is constantly attended by inflammatory changes, but does it originate in inflammation? Nearly five and twenty years ago, Sir William Bowman wrote: "Glaucoma is in its essence not an inflammation, and, when inflammatory, only so as it were by accident or complication."[1] To my mind, acute glaucoma is just as much an inflammatory disease as strangulated hernia, and no more. There is, I think, considerable likeness between these two diseases, and I am told that the late Mr. George Critchett was in the habit of drawing the same comparison. In the displacement of the bowel we have a condition, mechanical in its origin, which, for a long period of time, may have no serious consequences, but which may at its very outset or at any later time, under a slight constriction, be transformed into one of

[1] R. Lond. Ophth. Hosp. Reports, vol. v, p. 10.

acute and dangerous strangulation, with intense engorgement of vessels and outpouring of serum in the directions of least resistance. In the eye threatened by glaucoma we have an unfortunate relation of parts, which, though not itself a disease, may lead, through a little further encroachment upon the already narrowed space, to one of the most formidable of ocular disorders, involving stoppage of the intra-ocular currents, throttling of the circulation, and escape of the serum into the transparent media and the conjunctiva. Just as the taxis will occasionally remedy the displacement and terminate the danger in the one case, so in a few instances will eserine reopen the outlets and relieve the tension in the other; but as in strangulated hernia it is generally necessary to at once relieve the constriction with the knife, so in most cases of acute glaucoma our only means of cure is to promptly unlock the eye by iridectomy.

Now some pathologists, finding in eyes blinded by glaucoma, various inflammatory changes, exudation of leucocytes, cell-proliferation, adhesion between adjacent surfaces, and vascular changes, declare that inflammation is the starting point of the malady. It would, I think, be as reasonable to say that the exudations, the false membranes and the adhesions which are found in the sac of a hernia prove an inflammatory origin for that disorder.

Phlebitis affecting the vortex veins in their course through the sclera has been described as a cause of primary glaucoma.[1] I have examined these veins in many of my specimens, and in some of them have found the changes in question. Unfortunately, the microscope will not tell us at what stage of the disease they begin, but to me it seems more probable that they are consequences than that they are initial causes of the high pressure. A primary phlebitis, even if it began in the vortex veins, would surely extend beyond them, and its consequences would hardly be remediable by any operation. Moreover, these changes in the veins are to be found in secondary glau-

[1]Birnbacher and Czermak, Von Graefe's Archiv., vol. xxxii, Part ii, p. 1

coma also, and here they are certainly not the initial causes.

Inflammations may doubtless be the starting point of primary glaucoma in some instances, but there is no evidence, I think, that it is the usual and essential cause. The way in which the premonitions of glaucoma come and go, the fact, a drop of atropine can excite a violent attack in an eye which had previously shown no signs of inflammation, and that a surgical operation permanently can put an end to the whole process, are strongly opposed to the doctrine that glaucoma is essentially an inflammation.

Accommodative strain is believed by some observers to be a potent exciter of glaucoma.[1] Probably it may sometimes act by congesting the ciliary processes, but it must be remembered that the liability to glaucoma is greatest at a time of life when the accommodation is in abeyance. Much book-work is likely to be injurious, quite apart from the accommodative act, by reason of the stooping and the congestion of the head which it often involves.

Atropine, homatropine, duboisine, cocaine, in short, all the drugs which dilate the pupil, must be included amongst the exciting causes of primary glaucoma. It is easy to see that when the filtration angle is already dangerously narrow, the thickening of the iris-base which accompanies dilatation of the pupil may suffice to complete the blockade.

Finally, in connection with the causation of primary glaucoma, it is interesting to compare the liability of the two sexes as shown by the statistics already given. Men appear to be chiefly liable to the non-congestive, females to the congestive, forms, and the latter seem to be on the whole rather more liable than the former. Probably it would be more correct to say that while the liability to the simply non-congestive form is about equal in the two sexes, women are much more prone than men to congestive exacerbations, or even to anticipate the onset of the chronic disease by attacks of the congestive kind. This special tendency may reasonably be referred to

[1] Walker, Trans. Internat. Med. Cong., 1881, Sect. Ophthal., p. 88, Schoen, Trans. ternat. Ophth. Cong., 1888, p. 257.

the greater instability of the vaso-motor system in women, and particularly to the disturbances of circulation which emanate from the generative organs. So far as regards the predisposition which depends on the build of the eye and the growth of the lens, men and women probably stand on the same footing, but as regards the exciting causes women have the disadvantage.

The causes of primary glaucoma, then, are various, and not yet completely known; but they are, I think alike in this, that they all lead to compression of the filtration angle. With that compression the glaucoma process begins. The escape of fluid is retarded and the intra-ocular pressure rises; the increasing pressure hinders the flow of blood through the choroidal veins, and aggravates the swelling of the ciliary processes; this, in its turn, increases the compression of the filtration angle. Moreover, the blocking of the circumlental space checks the escape of surplus fluid from the vitreous chamber, and the lens, pressing forward, intensifies the mischief. And, further, if any fluid still exudes from the turgid ciliary processes, it is albuminous and less diffusible than the normal secretion. Thus cause and effect react upon each other in a vicious circle, and the glaucoma intensifies itself.

In acute glaucoma we see the vascular element at its maximum, in chronic glaucoma at its minimum; in the subacute and intermittent forms it seems to ebb and flow under various influences which aid or embarrass the circulation in the uveal tract. The vascular element finds its complement and auxiliary (more marked in some cases, less marked in others) in the predisposing structural condition of the eye.

It would carry us far beyond the limits of these lectures to discuss the consequences of an increase of the intra-ocular pressure. They constitute, for the most part, the well-known symptoms of glaucoma. They form a complex group of changes resulting from obstructed circulation, suppressed secretion, and distention and yielding of the tunics.

It will be well, in conclusion, to inquire how far our present knowledge of the pathology of glaucoma accords with the

rules and methods of treatment which have been established by clinical experience.

Principles of Treatment.—If it be true that an increase of the pressure within the eye is the essence of the glaucoma process, a reduction of this pressure must be the essential object of our treatment. Experience shows that the only measures which retard or arrest the process are those which reduce the tension of the eye.

Glaucoma usually calls for operative treatment, for the due escape of the intra-ocular fluid can seldom be permanently re-established by any other means, but there are some cases which can be successfully treated without operation, and there are very many in which certain auxiliary measures are of great value.

Eserine sometimes rapidly relieves the high tension. It is the antogonist of atropine which, as we have seen, sometimes induces or aggravates high tension. Other myotics and mydriatics have similar effects. It is important to notice that these drugs cause no decided changes of tension in the healthy eye, and that such small changes as they do produce are usually the opposites of those in question, eserine tending to raise, and atropine to lower, the intra-ocular pressure. Their action in glaucoma, therefore, must depend on some abnormality in the glaucomatous eye. This obnormality lies unquestionably in the altered relations of the iris.

Eserine, by contracting the sphincter of the pupil, thins the iris, flattens its folds and pulls upon its peripheral insertion. If the filtration angle is compressed it tends to reopen it. Accordingly, we find that eserine is chiefly useful when this compression is slight or recent. In the sudden, but comparatively mild, attacks which come and go during the premonitory stage of primary glaucoma, it acts with admirable effect. In severe acute attacks, also, it may be useful if applied without delay; but in such cases the sphincter of the pupil is soon paralyzed, and the filtration angle is very firmly closed, hence the period during which it can give relief is soon past. In chronic, non-congestive glaucoma, eserine often lowers the tension for a time, but the improvement is seldom great or lasting.

In some forms of secondary glaucoma also—for example in that which sometimes follows hæmorrhage into the vitreous and that which is caused by lateral displacement of the lens—eserine sometimes acts just as in primary glaucoma.

In all forms of the disease in which it is unable to contract the pupil, and thereby to increase the patency of the filtration angle, eserine is useless. When it is useless it is certainly harmful, for it increases the hyperæmia and often causes pain. On this account the strength of the preparation employed and the frequency of its application should be the minimum which suffices to contract the pupil and to keep it contracted.

In one form of secondary glaucoma, namely, that due to the presence of the lens in the anterior chamber, eserine has been known to act as the immediate cause of the attack by occluding the pupil and thus leading to closure of the filtration angle, the very complication which, in the reversed position of the parts, it is often able to relieve—a striking proof that its effects depend upon the position of the iris. Eserine, therefore, is not a specific for high tension, but simply a means of combating a particular displacement of the iris, which is often, but not always, the immediate cause of high tension.

Atropine is harmful precisely in those conditions in which eserine is useful; it induces high tension by dilating the pupil, slackening and throwing the iris into folds, and thereby helping to cbstruct the filtration angle. When it cannot dilate the pupil it never, I think, raises the tension. In certain cases of secondary glaucoma in which the high tension is due to serous exudation and not to displacement of the iris, atropine, by subduing the inflammation, tends to restore the normal tension. This is not mere hypothesis. A lady had severe secondary glaucoma from circular posterior synechia, and was iridectomized in both eyes with good result. Nearly a year later she came under my care with recurrence of injection and high tension in one eye; she had been strictly warned by two oculists against the use of atropine; nevertheless, and against her inclination, I ventured to prescribe it, and the injection and tension quickly subsided.

Cocaine, like every other dilator of the pupil, has been known to induce glaucoma. On the other hand, this drug has the power, invaluable in glaucoma, of contracting the ciliary blood-vessels and diminishing the sensibility of the ciliary nerves. By combining cocaine with eserine in such proportions that the eserine shall have the mastery over the pupil, we get the advantages of both without the disadvantages. Whenever eserine is used in the treatment of glaucoma it should, I think, be combined with cocaine.

Morphine will sometimes cut short, and will often alleviate a glaucomatous attack; it acts probably by easing pain, lowering the blood-pressure, and at the same time promoting contraction of the pupil.

Sleep, even though of a very short duration, often dispels the mild premonitory attacks with which primary glaucoma begins. During sleep the pressure in the cerebral vessels falls, and the pupil contracts.

Warmth, food, and rest relieve, just as cold, hunger, and fatigue induce, these early and slight attacks.

These palliative measures, though often very useful, have seldom more than a transient effect, and any discussion of them would be likely to do more harm than good, if it were to obscure the fact that the true remedy for glaucoma in the great majority of cases is a timely iridectomy.

Iridectomy, as performed for the relief of glaucoma, consists in the formation of an incision which opens the anterior chamber very near to its periphery, together with the excision of the corresponding segment of the iris. Von Graefe, to whom we are indebted for this invaluable remedy, was unable to explain its mode of action, yet every detail which he laid down with regard to its performance is sanctioned and confirmed by the more advanced pathology of the present day.

Iridectomy appears to effect the cure of glaucoma in the following way: In the first place, the escape of the aqueous humor and, when there is much congestion, the escape of blood, immediately lowers the pressure in the chambers. This, in the next place, relieves the obstructed circulation. The turgid

ciliary processes subside, fluid drains away from the vitreous through the circumlental space, and the filtration angle reopens. Lastly, if the filtration angle reopens insufficiently, the lips of the wound, especially the inner lips, remain imperfectly united, and an abnormal outlet for the intra-ocular fluid is permanently established. The absence of a segment of the iris and the presence of a more or less permeable cicatrix are safeguards for the future.

With regard to the circumlental space, since iridectomy cannot alter the size of the lens, its efficiency must depend chiefly on subsidence of the ciliary processes, and must, therefore, be greater in the more congestive forms of the disease. With regard to the filtration angle, a complete reopening is more likely to occur when the compression is still recent than when it has lasted many days or weeks. Accordingly we find that iridectomy acts with the greatest certainty in acute and recent cases. In the two eyes examined by Fuchs, which had been permanently cured of acute glaucoma by iridectomy, the filtration angle was patent throughout the whole circle.

In chronic non-congestive glaucoma, iridectomy acts with less certainty. When it permanently lowers the tension it appears to do so rather by creating fresh outlets than by restoring the normal ones. During the healing process the fluid appears to find for itself new filtration channels in the cicatrix and the adjacent tissues. The lips of the wound do not come into close apposition, but are joined by a thin semi-transparent tissue, and the overlying conjunctiva remains slightly elevated by the fluid which escapes into it.

In non-glaucomatous eyes, iridectomy causes no permanent reduction of the tension, and in the eyes of animals it has been found to diminish rather than increase the patency of the filtration angle.[1] On this ground some writers have maintained that it cannot promote filtration in the glaucomatous eye. But what is the behavior of artificial openings and false passages in other parts of the body—for example, in the urinary organs,

[1]Schoeler, Trans. Internat. Med. Cong., Sect. Ophth., p. 100; see Ophth. Rev., vol i, p. 216.

the intestine, and the lachrymal sac? If the normal outlet is patent, the artificial opening closes rapidly and completely; if the normal outlet is closed, a vicarious channel is permanently established. The same rule applies to the eye.

In some cases of glaucoma, iridectomy fails. We get neither a reopening of the normal outlets nor the establishment of new ones. The failure may arise in several ways. The lens may be injured, and may swell up and occlude the wound. It may do the same thing through displacement forwards under excessive pressure from behind. The incision may lie too far from the periphery of the chamber. A purely corneal cicatrix appears to admit of little or no filtration.

Sclerotomy, whether performed with the linear knife according to De Wecker's method, or with the keratome as advocated by Snellen and others, aims at opening the anterior chamber near to its periphery, and leaving the iris intact. Its success in many cases proves that the essential part of an iridectomy is the incision of the sclera, not the excision of the iris; it does not prove, however, that the latter step is better omitted. The disadvantage of sclerotomy is that during the healing process the iris is apt to occlude the wound; in congestive glaucoma it has, I think, the further disadvantage that it does not, like iridectomy, immediately promote an escape of blood from the turgid vessels.. It is, however, no part of my present purpose to discuss the details of these operations.

Artificial openings in other situations, for instance incisions made transversely through the ciliary region, and incisions which open the vitreous chamber only, though sometimes successful,are less to be trusted than a well-made iridectomy. It would appear that a filtration scar is more readily established in close proximity to the normal outlets than in any other part of the tunics.

Finally, from the pathological point of view, it might appear that primary glaucoma could be radically cured in its early stage by the extraction of the lens in its capsule, but the practical surgeon would shrink from attempting an extraction under such circumstances. The lens being healthy, would demand a

larger incision than is required for the cataractous lenses which we commonly extract, and it would separate from its attachments with great difficulty. The fact is interesting, however, that the intentional removal of the lens or its accidental escape after an iridectomy has proved distinctly beneficial in certain cases of glaucoma.

The treatment of glaucoma should not nowadays, I think, be called empirical. We know to a large extent the causes and nature of the morbid process, and we know the action of the measures by which we attempt to arrest it. It may be that our better knowledge of the causes will not supply us with better remedies than those which we now employ, but it will certainly enable us to employ them with more discrimination, more confidence, and more success.—*British Med. Jour.*

CORRESPONDENCE.

DEAR DR.—I have started a new Institution, which is named the New Amsterdam Eye and Ear Hospital, and situated at 212 W. 38th St., New York City. The Hospital was incorporated in April, 1888 and opened for the reception of Dispensary cases in June, since which time we have treated 700 patients and made 50 operations. The in-door department was opened in October. Our Board of Trustees consists of wealthy and well-known men, and I am the Surgeon-in-Chief.

Yours truly,

THOS. R. POOLEY.

NEW YORK CITY.

EDITORIAL NOTICE.

CONVENTION FOR THE REVISION AND PUBLICATION OF THE PHARMACOPŒIA OF THE UNITED STATES OF AMERICA.

To the Editors of all Journals of Medicine and Pharmacy throughout the United States of America.

It is suggested that you will have the kindness to call attention, in your editorial columns, to the call for a General Convention to assemble in Washington, D. C., at noon of May 7th, 1890, for the purpose of providing for a Revision and Publication of the Pharmacopœia of the United States of America; and that you will also request that every incorporated medical or pharmacal College, Association or Society desiring to be represented in the Convention will send to me its Corporate Title and a List of its Officers, addressed to care of Dr. Edwin H. Brigham, Assistant Librarian of the Boston Medical Library 19 Boylston Place, Boston, Mass., in order that I may prepare for publication, as directed by the Convention of 1880, a list of the bodies to be represented. Very respectfully,

ROBERT AMORY,

Boston, Mass., March 9, 1889. Pres't of the Conv'n of 1880.

Notice is hereby given that, in accordance with and by virtue of the authority vested in me by the Convention of 1880, I hereby call upon the several incorporated Medical Societies, incorporated Medical Colleges, incorporated Colleges of Pharmacy, and incorporated Pharmaceutical Societies throughout the United States, The American Medical Association and the American Pharmaceutical Association to elect a number of delegates, not exceeding three, and upon the Surgeon-General of the Army, Surgeon-General of the Navy and the Surgeon-General of the Marine Hospital to appoint, each, not

exceeding three medical officers to attend a General Convention for the Revision and Publication of the Pharmacopœia of the United States of America, to assemble in the City of Washington, D. C., on the first Wednesday of May, 1890 (May 7th), at 12 o'clock noon.

The several bodies, as well as the Medical Departments of the Army, Navy and Marine Hospital Service, are hereby requested to submit the Pharmacopœia to a careful revision, and to transmit the result of their labors to the Committee of Revision at least three months before the meeting of the General Convention.

The several Medical and Pharmaceutical bodies are hereby requested to transmit to me, as the President of the Convention of 1880, the names and residences of their respective delegates as soon as they shall have been appointed; a list of these delegates shall thereupon be published under my authority, for the information of the medical public, in the newspapers and medical journals in the month of March, 1890.

In the event of the death, resignation or inability of the President of the Convention of 1880 to act, these duties (in accordance with the resolution of that convention) shall devolve, successively, in the following order of precedence, upon the Vice-Presidents, the Secretary, the Assistant Secretary, and the Chairman of the Committee of Revision and Publication of the Pharmacopœia.

These officers are as follows: First Vice-President, Samuel C. Busey, M.D., of Washington, D. C.; Second Vice-President, P. W. Bedford, Ph.G., of New York; Secretary, Frederick A. Castle, M.D., of New York; Assistant Secretary, C. H. A. Kleinschmidt, M.D., of Washington, D. C.; Chairman of Committee of Revisision, Charles Rice, Ph.D., of New York; First Vice-Chairman of the Committee of Revision, Joseph P. Remington, Ph.M., of Philadelphia, Pa.; Second Vice-Chairman of the Committee of Revision, C. Lewis Diehl, Ph.G., of Louisville, Ky.

At the general convention held in Washington, D. C., on the fifth day of May, 1880, the organizations and bodies

enumerated in the Abstract of the Proceedings of the National Convention of 1880, on pp. xv. to xviii. of the United States Pharmacopœia of 1882—a list of which will be found appended to this call—were recognized as being entitled to representation.

If any body other than those admitted in 1880 shall desire a representation in the convention of 1890, it is suggested that the proof of incorporation, signed by the Secretary of State, of the State which shall have issued the charter, or by properly qualified public officials of the United States, be presented with the credentials of the delegation.

A blank form of certificate of appointment of delegates will be sent upon application by letter or to my address, care of Dr. Edwin H. Brigham, Assistant Librarian of the Boston Medical Library, 19 Boylston Place, Boston, Mass.

(Signed.) ROBERT AMORY,
President of the Convention of 1880.

Boston, March 9, 1889.

LIST OF INCORPORATED BODIES AND OF GOVERNMENT DEPARTMENTS REPRESENTED IN THE PHARMACOPŒIAL CONVENTION OF 1880.

Connecticut Medical Society.
Iowa State Medical Society.
Massachusetts Medical Society.
Medical Society of the State of New York.
Medical Society of the State of North Carolina.
College of Physicians and Surgeons in the City of New York.
College of Physicians, Philadelphia.
Medical and Chirurgical Faculty of Maryland.
Medical Society of the District of Columbia.
New York Academy of Medicine.
Philadelphia County Medical Society.
Albany Medical College, Med. Dept. of Union University, Albany, N. Y.
Bellevue Hospital Medical College, New York.

College of Medicine, Syracuse University, Syracuse, N. Y.

College of Physicians and Surgeons, Med. Dept. of Columbia College, New York.

Dartsmouth Medical College, Hanover, N. H.

Department of Medicine and Surgery of the University of Michigan, Ann Arbor.

Jefferson Medical College, Philadelphia, Pa.

Medical College of Indiana, Med. Dept. of Butler University, Irving, Ind.

Medical Department of Howard University, Washington, D. C.

Medical Department of Iowa State University, Iowa City.

Medical Department of the University of Georgetown, Washington, D. C.

Medical Department of the University of Maryland, Baltimore.

Medical Department of the University of Pennsylvania, Philadelphia, Pa.

Medical Department of the University of Virginia, Charlotteville, Va.

Miami Medical College, Cincinnati, O.

Missouri Medical College, St. Louis.

National Medical College, Med. Dept. of Columbia University, Washington, D. C.

Rush Medical College, Chicago.

University of the City of New York, Med. Dept., New York.

Woman's Medical College of the New York Infirmary, New York.

Women's Medical College of Pennsylvania, Philadelphia, Pa.

Chicago College of Pharmacy, Chicago, Ill.

Cincinnati College of Pharmacy, Cincinnati, Ohio.

College of Pharmacy of the City of New York, N. Y.

Louisville College of Pharmacy, Louisville, Ky.

Maryland College of Pharmacy, Baltimore, Md.

Massachusetts College of Pharmacy, Boston, Mass.

National College of Pharmacy, Washington, D. C.

Pennsylvania College of Pharmacy, Philadelphia, Pa.
Philadelphia College of Pharmacy, Philadelphia, Pa.
St. Louis College of Pharmacy, St. Louis, Mo.
University of Michigan, School of Pharmacy, Ann Arbor, Mich.
Medical Department of the U. S. Army.
Medical Department of the U. S. Navy.
U. S. Marine Hospital Service.